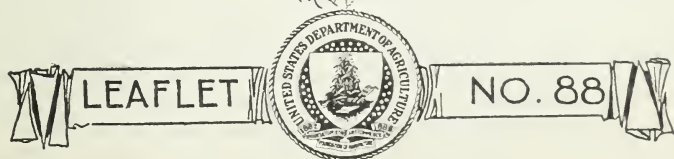


Historic, archived document

Do not assume content reflects current scientific knowledge, policies, or practices.

POISONING OF LIVESTOCK

BY PLANTS THAT PRODUCE
HYDROCYANIC ACID



POISONING OF LIVESTOCK BY PLANTS THAT PRODUCE HYDROCYANIC ACID

By JAMES F. COUCH,¹ chemist, Pathological Division, Bureau of Animal Industry

Many species of both wild and cultivated plants in the United States are capable, under certain circumstances, of developing hydrocyanic acid, also called prussic acid, which is highly poisonous. Such plants are known as cyanogenetic. Each year many inquiries about the possibility of livestock being poisoned by these plants are received by the United States Department of Agriculture. The following facts will assist livestock owners in recognizing the danger when it exists and in avoiding it.

Cyanogenetic Plants Fatal to Animals

A great many plants are capable of developing hydrocyanic acid and may be responsible for poisoning, but under practical conditions only a few are actually dangerous. A list of those most dangerous by reason of their poisonous character and wide distribution is given below. One or more of these plants is found in practically every part of the United States.

Chokecherry (wild), *Prunus virginiana* L.
Black cherry (wild), *Prunus serotina* Ehrh.
Sorghum, *Sorghum vulgare* Pers.
Johnson grass, *Sorghum halepense* (L.) Pers.
Flax, *Linum usitatissimum* L.
Arrowgrass, *Triglochin maritima* L. and *T. palustris* L.
Sudan grass, *Sorghum vulgare* var. *sudanense* (Piper) Hitchc.
California desert almond, *Emplectocladus fasciculatus* Torr.
Prairie flax, *Linum lewisii* Pursh.
Queen's root, *Stillingia dentata* (Torr.) Britton and Brown.

Factors Affecting Quantity of Hydrocyanic Acid in Plants

Free hydrocyanic acid as such is not found in any appreciable quantities in healthy growing plants. The acid develops when the normal growth of plants has been retarded or stopped by drought, frost, bruising, trampling, wilting, mowing, or other cause. Under such conditions hydrocyanic acid is formed by a chemical reaction between two substances, a glucoside and an enzyme, contained in the plant. Neither of these substances is poisonous by itself, and under normal conditions they are not in contact with each other. When cyanogenetic plants are eaten by animals, the plant tissues are crushed, and the glucoside and enzyme mix in the juice and in the moisture of the animal's stomach or rumen. Under such conditions hydrocyanic acid may be produced.

The quantity of potential hydrocyanic acid that can be formed in plants may vary considerably with the stage of growth, climatic conditions, and soil. In general, mature cyanogenetic plants contain a smaller percentage of potential acid than do young plants. Young sorghum suckers are about as poisonous as young first-growth plants; young second-growth plants are often more dangerous than young first-growth plants. Plants grown on poor soil contain less of the

¹ Resigned January 1940. Slightly revised by R. R. Briese.

acid than those on good soil, and fertilization with nitrates markedly increases the percentage of the acid. Sorghums grown in Southern States have not poisoned livestock so much as those grown farther north. The reason for the difference is probably climatic, but little is known about the exact causes of the formation of the poisonous acid in this case.

Dried cyanogenetic plants, such as those made into hay, commonly produce very much less hydrocyanic acid than plants which are fresh. Well-cured sorghum and Sudan grass are usually safe to feed, but occasionally they contain enough hydrocyanic acid to cause poisoning. It is thought that in some cases the poisonous acid is slowly given off during the process of drying and, being very volatile, passes into the air. In other cases some chemical alteration destroys the ability of plants to produce the acid. Some plants, however, retain a considerable proportion of the active acid even when dried. This applies especially to seeds. Dry immature flaxseed has caused hydrocyanic-acid poisoning. Dry rangoon beans, bitter almonds, peach kernels, and wild-cherry bark contain much potential hydrocyanic acid.

Preventive Measures Against Poisoning

Even when they contain considerable quantities of potential hydrocyanic acid, the plants may not poison livestock. Since poisoning depends upon the presence of actual hydrocyanic acid, anything that prevents its development in the stomach lessens or entirely removes the danger of poisoning. Certain feeds, such as alfalfa hay and linseed cake, retard the production of hydrocyanic acid and may prevent poisoning. Glucose in the paunch markedly represses the rate of formation of the acid. It has been observed that cattle on a corn ration are less likely to be poisoned when grazing on dangerous sorghums than are cattle not fed corn. It is therefore a wise precaution to give animals a starchy feed, such as corn, milo, or feterita, before allowing them to graze in the vicinity of plants capable of developing hydrocyanic acid.

An alkaline condition of the paunch also diminishes the rate at which the poisonous acid forms, and may even convert it into harmless products. But ordinarily it is not possible to bring about this alkaline condition as a preventive measure. If one animal shows symptoms of hydrocyanic-acid poisoning, other animals should be removed immediately from the pasture. It is commonly and probably correctly believed that the most dangerous plants are those that have wilted and in which hydrocyanic acid is already formed. When an animal eats wilted cherry leaves, for instance, it receives ready formed a dose of the poison. When normal, unwilted cherry leaves are eaten, the hydrocyanic acid must develop in the animal's stomach and be subject to the repressive influences previously mentioned. This accounts for some cases of poisoning from wild-cherry leaves that have not been otherwise satisfactorily explained.

Symptoms of Hydrocyanic-Acid Poisoning in Livestock

Hydrocyanic acid acts very rapidly, frequently killing the animal within a few minutes, although sometimes the poisoned animal may live for several hours after the symptoms develop. There is first

a brief period of stimulation followed by depression and paralysis. Symptoms of colic often appear. Stupor, difficult breathing, and frequent convulsions result from the action of the poison on the brain centers that control respiration. Death is caused by respiratory paralysis, the heart continuing to beat for some time after breathing has stopped.

Quantity Sufficient to Cause Sickness Usually Fatal

If the dose is sufficient to sicken an animal it is highly probable that the animal will die. Six grains (0.4 gram) of pure acid is considered a fatal dose for the average horse. Recent work by this Bureau indicates that a 1,000-pound cow would be made sick by 6 grains (0.882 milligram per kilogram) and killed by 14.2 grains (2.042 milligrams per kilogram), and that a 100-pound sheep would be made sick by 0.69 grain (0.992 milligram per kilogram) and killed by 1.61 grains (2.315 milligrams per kilogram). A 20-pound dog may be expected to die if given 0.03 to 0.04 gram, which is one-tenth the lethal dose for horses.

If a plant, then, contains as little as 0.02 percent potential hydrocyanic acid, and if the animal consumes it all rapidly, 5 pounds of the plant would be fatal for a horse or cow and 1.25 pounds would kill a sheep, if no factors entered in to prevent the development of the acid. Under actual conditions, however, on account of interfering factors such as those already considered, those quantities will not always prove fatal. But since cyanogenetic plants may contain 10 to 20 times as much potential acid as in the case just cited, it becomes evident that the fatal dose of these plants may be comparatively small and the danger to livestock correspondingly great.

Remedies

Recent studies by a number of investigators have developed new methods that are very promising for treating cyanide poisoning. The injection of methylene blue, sodium nitrite, or sodium thiosulphate, preferably intravenously, has proved to be a practicable and valuable procedure. Especially promising is the injection of a combination of sodium nitrite and sodium thiosulphate. For cattle, 2 to 3 grams of sodium nitrite in water followed by 4 to 6 grams of sodium thiosulphate in water has protected against two minimal lethal doses. For sheep, up to 1 gram of sodium nitrite and 2 to 3 grams of sodium thiosulphate are recommended. This treatment may be supplemented by other measures such as injections of atropine, inhalation of ammonia, injections of glucose, or symptomatic treatment. Since the use of these drugs is attended with some danger to an animal, a trained veterinarian should conduct or supervise the treatment.

